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Canine mast cell tumors express stem cell **factor receptor.**
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c-kit protooncogene encodes a type III transmembrane receptor kinase, the **stem cell** factor receptor, or KIT. The ligand of the KIT. **stem cell** factor, is a cytokine that stimulates mast cell growth and differentiation. We have studied immunohistochemically KIT expression in 23 canine mast cell tumors (MCTs), 10 histiocytomas, 5 malignant melanomas, and in 2 cell lines derived from mast cells (HMC-1, human and C2, canine). As expected, KIT was detected both in the human mast cell leukemia cell line (HMC-) and in the canine mastocytoma cell line C2. In normal canine skin, KIT expression was confined to mast cells. All canine MCTs expressed KIT, although the intensity of the staining reaction varied considerably among the 23 neoplasms. Grade III tumors showed the highest expression of KIT, whereas grade I tumors showed the lowest expression of KIT. Two patterns of KIT expression were detected in mast cells. In normal canine mast cells and in some neoplastic mast cells, KIT appeared mainly on the cell membrane. However, in many canine MCTs, KIT is accumulated in the cytoplasm, usually near the cell nucleus. The meaning of these two patterns is not clear. Expression of KIT could not be detected immunohistochemically in any of the other neoplasias investigated. According to our results, it can be concluded that most, if not all, canine MCT express KIT. Furthermore, there is an inverse correlation between the degree of differentiation and the expression of KIT. Moreover, according to our results, KIT can be used as a reliable immunohistochemical **marker** for canine mast cells and undifferentiated mast cell tumors.

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Differential expression of lymphoid and myeloid markers on differentiating hematopoietic stem cells **in normal and tumor-bearing adult human liver.**

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The presence and phenotype of lineage-committed hematopoietic progenitors in the normal adult human liver (AHL) were investigated and compared with the profiles of differentiating hematopoietic precursor populations detected in liver bearing metastases of colonic origin. Levels of hematopoietic **stem cells** (HSCs) (CD34(+)CD45(+)) detected in hepatic mononuclear cell (HMNC) populations were increased 6-fold when compared with matched peripheral blood samples. In normal liver, less than 5% of HSCs expressed the myeloid-associated antigen, CD33, whereas considerable proportions expressed lymphoid-associated **markers** (T cell, 33.39%; B cell, 17.39%; and natural killer [NK] cell, 37.17%). Significant increases were observed in the relative proportions of hepatic HSCs coexpressing CD33 (20.53%; P =.001), and the T-cell **marker** (CD7, 58. 13%; P =.02) in tumor-bearing liver compared with normal liver. HSCs with B-cell progenitor phenotype (CD19(+)) were significantly decreased in tumor-bearing liver (0.06%; P =.02). Despite these differences, the activation status of hematopoiesis, as measured by the coexpression of the differentiation and activation **markers** , CD38 and CD45RA, did not differ significantly between normal and tumor-bearing liver. These results indicate that the normal AHL harbors lineage-committed hematopoietic progenitors, and the vast majority of these progenitors express lymphoid-associated antigens with changes occurring in both the myeloid and lymphoid compartments of the hepatic hematopoietic pathway on tumor challenge. While tumor-bearing livers are enriched for intrahepatic myeloid precursors and T-cell progenitor cells, further studies are required to establish the origin and in situ development potential of hepatic HSCs in the adult human and their role in tumor immunity.

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Specific features of stem cell **(CD34+) subvariant of acute lymphoblast cell leukemia in children]**

Osobennosti stvolovokletochnogo (CD34+) podvarianta ostrogo limfoblastnogo lekoza u detei.

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Document type: JOURNAL ARTICLE ; English Abstract

The authors have examined 134 children with acute lymphoblast cell leukemia (ALCL) who were treated at the Research Institute of Pediatric Oncology and Hematology, Russian Cancer Research Center, in January 1990 to November 1999, and followed up till March 1, 1999. The mean duration of follow-ups was 57.46 +/- 2.87 months. The minimum follow-up was 4 months. The immunophenotypical features of **stem cell** immunological subvariant of ALCL identified from the presence of 10% or 15% of CD34+ lymphoblast cells were similar in the leukemia clone. There is evidence for the isolation of the least mature, **stem -cell** immunological subvariant of ALCL in children. The immunophenotypic features of **stem -cell** ALCL in children were as follows: the expression of myeloid antigens (linear and different **marker** leukemias) and the higher rates of involvement of B-cell leukemias. The clinical and hematological features of **stem -cell** versus CD34-negative ALCL in children were the low levels of leukocytes ($p = 0.009$) and blast cells ($p = 0.018$) in the peripheral blood at diagnosis. At the same time, **stem - cell** ALCL showed a poorer prognosis. Moreover, blast cell CD34 antigen expression deteriorated prognosis for pre-pre-B (common) immunological variant of ALCL ($p = 0.035$). Intensified ALCL treatment programmes improved relapseless survival of patients with **stem - cell** ALCL ($p = 0.0042$).